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Case Report

An unusual circumstance of internal chemical burn injury – A case report

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Abstract

Chemical burn injuries including nitric acid injuries are rarely seen in routine clinical practice. In this article, we describe a case of chemical burns due to ingestion of nitric acid in which the history was not of an accidental but of a suicidal nature. Spillage of nitric acid (vitriolage) is frequently reported especially in the third-world countries, but an ingestion injury like this is uncommon and rarely reported.

Keywords: Chemical burn injury; Nitric acid; Internal autopsy findings in nitric acid ingestion

Introduction

Nitric acid, also known as aqua fortis (strong water) or spirit of nitre or engraver's acid[1] is a chemical important for industrial and domestic purposes. A strong acid, powerful oxidizing agent and an ability to nitrate organic material make it an essential in the production of numerous chemicals. Skin contact leads to severe burns and its vapours can cause severe acid burns to the eyes, respiratory tract, and lungs. Being a corrosive, it produces immediate pain and causes burns of mouth, throat, esophagus and abdomen, widespread gastroenteritis, and bloody diarrhoea. Blood may also be found in urine. Unlike sulphuric acid, when concentrated nitric acid is ingested, the tendency to produce charring of tissues and then perforation is a rare event as recorded in the present case. The present case describes the macroscopic findings and may be helpful in formulating an emergency treatment protocol.

Case report

A 55-year-old female unable to face the problems of life, ingested an acid around 11 a.m. in the morning. She was brought with complaints of pain and burning sensation and thereby was admitted to a private medical hospital around 3.00 p.m. the same afternoon. She was a known diabetic. Following admission, the patient had undergone laboratory investigations which revealed red colored urine (haematuria), proteinuria, aciduria (low urine pH), and pyuria suggesting signs of poisoning and later septic shock. Amorphous calcium oxalate crystals were also found in urine. Serum electrolytes and other routine investigations were normal. Liver function tests (LFT) showed raised liver enzymes (SGOT = 91, SGPT = 46). Peripheral smear showed a total count of 22,300 (N_{92} L_7 M_1) which is a sign of acute inflammation and perforation. This was later confirmed with an abdominal X-ray showing pneumo-peritoneum. The patient's condition deteriorated after 2 hours. Arterial blood gas analysis showed acidosis with a pH of 7.1. Serum electrolytes showed variation (serum potas-

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sium = 5.2, and base deficit = −20.7). Surgical opinion was sought but it was deferred till her general condition improved. To stabilize this, she was started on IV fluids and bicarbonates. At 10 p.m. when she suddenly developed bradycardia following which she was further treated with vasopressors and antibiotics. All these measures were in vain and patient succumbed to death. In view of the unnatural cause of her death, a postmortem examination was conducted at the District Wenlock Hospital, Mangalore, to shed light to the cause of death.

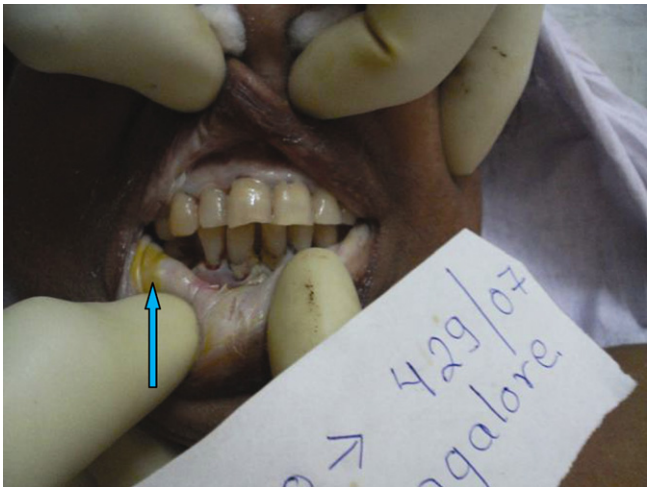

Fig. 1. Oral mucosa showed yellowish discolouration (xanthoproteic reaction).

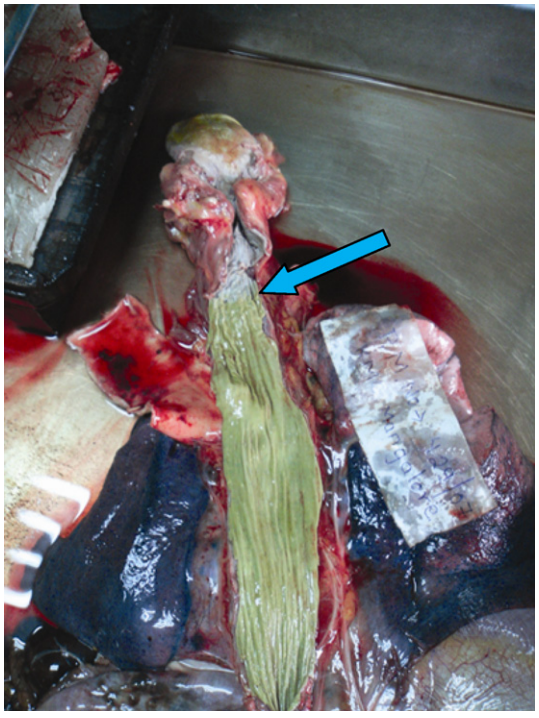

Fig. 2. Lingual and oesophageal mucosa showed yellowish discolouration.

Postmortem findings showed a body of an adult female with wheatish complexion aged 55 years, moderately built and nourished, measured 152 cm and weighed 65 kgs. Yellowish discoloration (Fig. 1) around the peri-oral mucosa

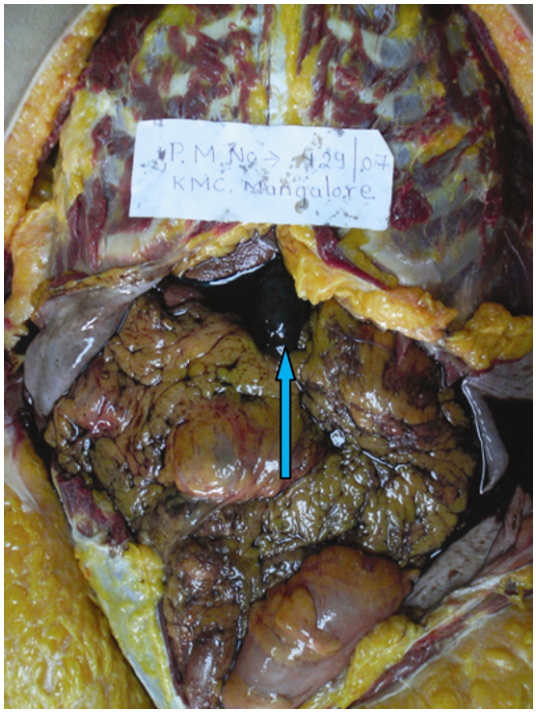

Fig. 3. Black colored acidic fluid in the peritoneal cavity due to perforated stomach.

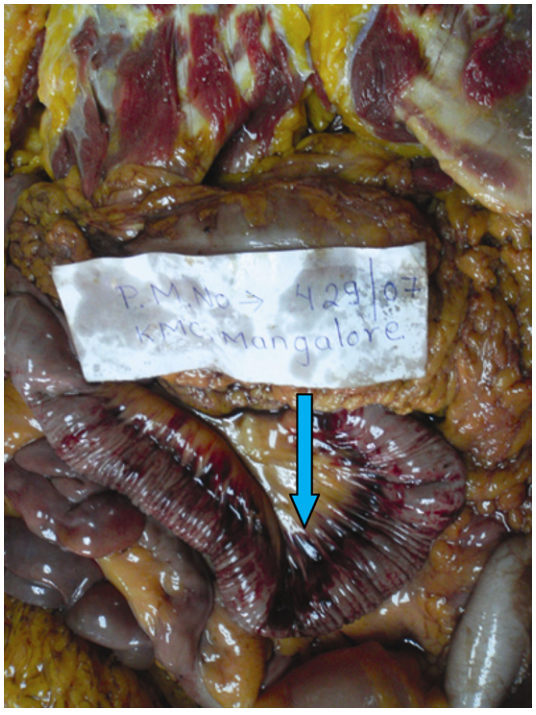

Fig. 4. Mesentery showed reddish discolouration all along their attachment to the intestines.

and whitish tinge of teeth was seen. The lingual surface (Fig. 2) (tongue) too showed yellowish discoloration due to denaturation. Peritoneum was filled with 300 ml of dark black (Fig. 3) colored fluid (burning sensation on touch). Stomach contained similar contents; mucosal surface was dark brown to black (Fig. 6) in color (charring) with a single perforation measuring (5 × 3 cm) in the pyloric region (Fig. 5). External surface of stomach and large intestine too showed dark black to brownish discoloration. Intestines showed similar contents too. Mesentery showed reddish discolouration all along their attachment to the intestines (Fig. 4). Organs were congested. Lungs, liver, heart and intestines showed hemorrhages.

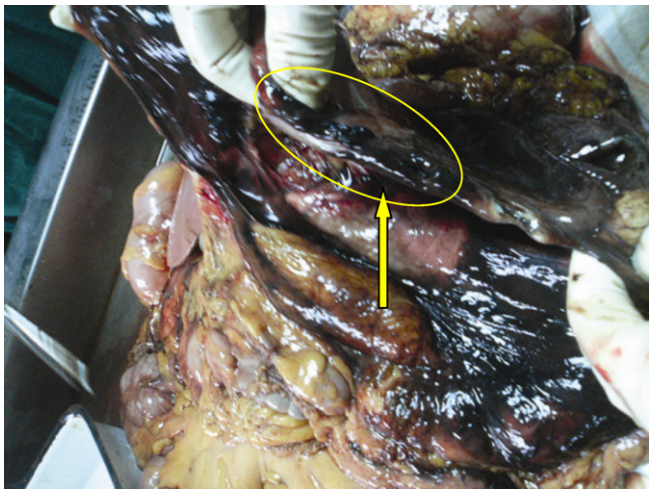

Fig. 5. Perforation measured (5 × 3 cm) in the pyloric region.

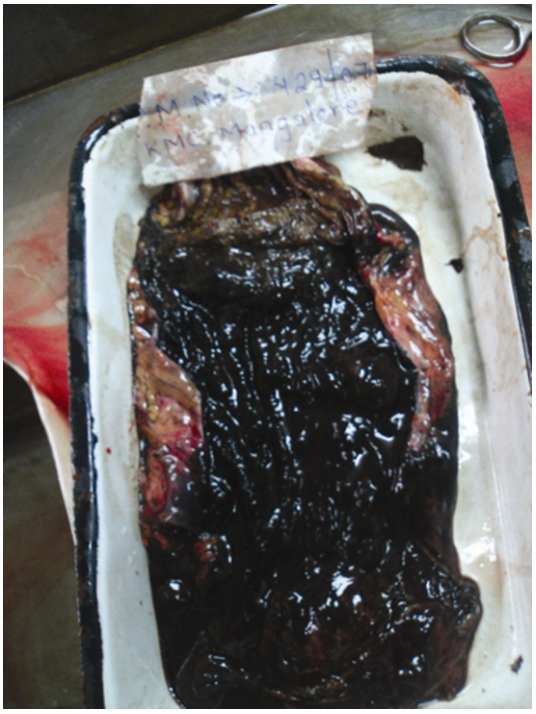

Fig. 6. Gastric mucosa showing charring.

Viscera sent for toxicological qualitative analysis showed the presence of a mineral acid, nitric acid pH 3.2, highly acidic in nature.

Cause of death

Perusal of forensic science laboratory and autopsy findings concluded that the deceased died due complications (chemical peritonitis) of consumption of nitric acid.

Discussion

Chemical burn injuries constitute a type of burn injury that we may come across in our daily practice, although not as frequently as thermal and electric injuries. Chemical agents involved are acids, alkalis or organic compounds. Acids such as sulphuric acid, nitric acid, hydrochloric acid, tannic acid and formic acid are widely used and handled by many individuals. However the potentially deleterious effects on human health of many of these chemicals are largely unknown. They are contained in bathroom cleaners and rust removers and are routinely used by automobile battery manufacturers, in the dyeing industry, in tanneries and in jewellery workshops. Although rare, occupationally and accidentally induced acid burns have been reported as in the literature.[2–4] Suicidal ingestion appears rare.

Most chemical agents damage the skin by producing a chemical reaction rather than hyperthermic injury. Nitric acid in contact with skin or mucosal surface produces yellow discolouration due to a reaction with the protein keratin (Xanthoproteic reaction) distinguishing it from poisoning by other acids. The pathophysiology depends on the type of the acid, its pH, its molar concentration, the volume ingested, duration of contact, the presence of other contents in the stomach and the gastric emptying time of the acids concerned.[1] Esophageal injury occurs in 6–20% of acid ingestion. Esophageal injury is mild as there is a rapid transit and limited permeability, hence less chances of perforation which will always be associated with gastric damage.[1] Acids cause coagulation necrosis of the tissues with thrombus formation in the microvasculature of the lesion. In high concentration and volume, nitric acid reacts with blood forming acid hematin, which is the cause for charring (black discolouration) as seen in this case. Rapid loss of collagen and mucopolysaccharides occur. If these acids are absorbed through the skin, they may cause systemic effects such as metabolic acidosis or renal failure as seen in present case. Death occurs from circulatory collapse or from secondary destructive changes in the gastrointestinal tract.

In all chemical injuries, the primary aim is the removal of the offending agent because the longer the offending agent is in contact with the skin or any other structure, the more severe the injury becomes. There are specific antidotes for certain chemical agent injuries but, whatever the agent, initial treatment consists of the removal of clothing (including underwear, gloves, and shoes) and irrigation with copious

amounts of water or saline. With regard to the kind of case that we presented here, apart from occupational and accidental injuries, acid burn injuries are usually planned as an assault on the victim for reasons of revenge. The victim is mostly a woman, males are relatively less affected. Common causes are love affairs, enmity, and personal reasons. The aim of the attack is not to kill, but to make victim live with a sequelae, a mark always be left to remember. The incidence is greater in rural than urban areas.

Conclusion

It may be said that acid burn injuries represent only a minute percentage of burns, but they cause a particular type of lesion in which the morbidity is high and death is certain. This case is an unusual circumstance of an internal burn injury caused due to nitric acid, a rare event, made more so by being used as an agent for suicide.

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